VOT voicing distinction is robust along a speech rate continuum in dysarthria

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**Background:** Approximately 50% - 90% of people with Parkinson’s disease (PD) eventually develop hypokinetic dysarthria [1], a motor speech disorder characterized by imprecise consonants, a quiet, monotonous voice, and fast rushes of speech [dab1969clusters]. One consequence of HkD is deviant stop consonant production compared to healthy, age-matched controls. For example, voice onset time (VOT), a robust stop voicing cue [2], may be abnormal in PD, though evidence does not point to a clear pattern in the directionality of abnormal production. While some studies have reported no differences in VOT between people with PD and healthy, age-matched controls [3–6], some reports of longer voiced VOT [7] and shorter voiceless VOT [8, 9] suggest that there may be a reduced voicing contrast for these talkers.

Targeting a slower rate of speech is a common behavioral intervention in speech therapy for some people with PD [10]. Slow speech is thought to allow talkers time to reach more target-like vocal tract configurations, potentially aiding in increasing acoustic clarity of their speech [11]. Recent work suggests that rate reduction does not lead to improvements in speech outcomes for many people with PD [12]. Furthermore, there is a small body of evidence that suggests faster speech is not always detrimental to intelligibility for some people with PD [13]. To date, most studies have explored a small number of speech rate adjustments for talkers with PD, and have not systematically explored the effect of rate on stop voicing distinctions. Rate studies in young, healthy talkers suggest that voiceless but not voiced VOT is highly sensitive to changes in speech rate [14], perhaps because doing so would reduce the voicing contrast [15]. It is not presently known what adjustments are made by talkers with HkD. The purpose of the present study was to elicit a broad continuum of speech rates from very slow to very fast to explore changes in VOT production in talkers with PD and healthy, older controls.

**Method:** Two English-speaking participant groups were included in the present study: people with PD (n = 34), and healthy, older controls (OC, n = 17). As part of a larger battery of speech tasks, all participants read aloud a list of 24 nonce words of the form “aCVd” embedded in a carrier phrase, where C was one of six stop consonants (/p/, /t/, /k/, /b/, /d/, /g/) and V was a corner vowel. All talkers participated a magnitude production task that elicited speech rates from very slow to very fast (later binned into a five-step continuum). Habitual rate was always elicited first. Modified rates were elicited in separate blocks and were counter-balanced, and were accompanied by a brief practice period. All stimuli were elicited within each rate condition in a random order. VOT was measured for all stops and modelled as a function of speech rate, group, and voicing using linear mixed-effects regression.

**Results and Discussion:** As demonstrated in Figure 1, At the slowest rate, the OC group
demonstrated a greater voiced-voiceless VOT distinction compared to the PD group (group-rate voicing interaction: \( p < 0.05 \)). Conversely, at faster rates, the OC group produced smaller VOT voicing contrasts compared to the PD group (\( p < 0.01 \)). Pairwise comparisons revealed that this difference was mainly driven by group differences in voiced VOT. That is, while all talkers modified the stop voicing contrast at extreme ends of the rate continuum, the overall contrast was less changed for the PD group, in large part to their unexpected lengthening of voiced VOT.

Overall, across a continuum from very slow to very fast, stop voicing distinctiveness increased in slower speech and decreased in faster speech, though to a lesser degree for the PD talkers. Findings suggest implications for clinical considerations of rate modification, as well as a clearer understanding of stop consonant abnormalities in PD.

References